# TREATMENT OF EXOPHTHALMOS AS VIEWED BY THE OPHTHALMOLOGIST

**Moynihan Lecture delivered at the Royal College of Surgeons of England**

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**by**

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WHEN ONE CONSIDERS that any increase in bulk within the orbit, or pressure of expanding lesions in the structures surrounding the orbit, pushes the globe forward in exophthalmos, the number of possible causes is amazing. Henderson (1956), in his investigation, lists seventy conditions producing exophthalmos. Obviously exophthalmos is not a disease entity but a cardinal symptom of a number of diseases and affections. It is necessary, for proper treatment, to know the cause, as treatment varies from temporising with periodic examination to immediate exenteration of the orbit. Knowledge of the type of lesion is essential, the location and size are of next importance.

To establish a diagnosis from such a variety is difficult but an attempt will be made to group the causes, as others have done, into some sort of organized form better to determine proper treatment.

The majority of conditions producing exophthalmos can be included in six categories.

## CAUSES OF EXOPHTHALMOS*

1. CONGENITAL AND HEREDITARY
   - (*a*) Structural deformities with shallow orbits
   - (*b*) Absence of orbital bone with cerebral herniation
2. INFLAMMATORY (INFECTIOUS)
   Cellulitis, abscess, granulation (Granulomata—pseudo-tumour, gumma, tuberculoma, etc.)
3. VASCULAR
   - (*a*) Venous engorgement, varix, thrombosis
   - (*b*) Arterial aneurysm, arteriovenous fistula
4. TRAUMATIC
   - (*a*) Oedema
   - (*b*) Haemorrhage
   - (*c*) Emphysema
5. METABOLIC
   - (*a*) Endocrine: thyrotoxic, thyrotropic
   - (*b*) Lipodystrophy—orbital deposits—diabetic exophthalmos

*Exophthalmos may be divided roughly into unilateral (local) and bilateral (systemic). It can be divided into inflammatory and non-inflammatory. But these classifications are too broad and loose to be very useful.

6. NEOPLASTIC—Benign and malignant tumours
   (*a*) Primary
   (*b*) Secondary (extension, invasion)
   (*c*) Metastatic

## PSEUDO-EXOPHTHALMOS

Congenital glaucoma
Myopia
Thyrotoxicosis (lid lag and retraction)
Facial paralysis
External ophthalmoplegia
Sympathetic irritation (Claude Bernard Synd.)
Cicatricial lid deformities

## EXAMINATION OF EXOPHTHALMOS

1. LOCATION
   (*a*) Unilateral—localized
   (*b*) Bilateral—systemic
2. POSITION
   (*a*) Axial—within muscle cone
   (*b*) Eccentric—without muscle cone
3. AMOUNT
   Measured with exophthalmometer (Drews, 1957)
4. REDUCIBILITY
   Possible with thyrotoxic and soft tumours
   Impossible with thyrotropic and hard tumours
5. CONSISTENCY
   Measured with orbitometer (N 20-62 mm. water—inconstant)
   Hard, soft, fluctuating
6. CONSTANCY (variation in size and amount)
   (*a*) At various periods
   (*b*) In various positions
   (*c*) With stress and strain
   (*d*) With compression of vessels of the neck or orbit
7. PULSATION AND BRUIT (vascular and cerebral)

## SYMPTOMS ACCOMPANYING EXOPHTHALMOS AND THEIR DETERMINATION

1. MOTILITY—INTERFERENCE (DIPLOPIA)
   Paresis or paralysis measured with deviometer (Ingalls, 1953)
2. REDUCTION OF VISION AND VISUAL FIELDS
   Ophthalmoscopic evidence
   (*a*) Papillary congestion, oedema, atrophy
   (*b*) Retinal striae or invasion
   Perimetric and tangent screen evidence
3. APPEARANCE
   (*a*) Oedema and chemosis
   (*b*) Engorgement and discolouration
4. SUBJECTIVE
   Discomfort, pain and tenderness (diplopia, reduced vision)
5. EXTRA OCULAR AND SYSTEMIC EVIDENCE
   Skin, central nervous system, nose and throat
   Lesions elsewhere (systemic or localized)

6. X-RAY
   Size or orbit
   State of orbital rim and bony orbit
   Size of fissures and optic foramen
   Soft tissue density (pneumogram)
   Vascular anomalies (angiogram-venogram)

LABORATORY TESTS
   BMR, Cholesterol, calcium, sugar, etc.
History of patient and family may be of great importance.

**Congenital and hereditary anomalies**

We will dwell briefly on congenital and hereditary anomalies which may account for both true and pseudo-exophthalmos. Exophthalmos may be the result of absence of a portion of the bony orbit through which meningocele and encephalocele herniate in which pulsation is an important symptom. Frequently neurofibroma is present, occasionally astrocytoma of the optic nerve. The same condition is occasionally experienced when as the result of a transfrontal operation considerable orbital bone is removed. This happens only when dura is thin or absent or intracranial pressure is increased. Hand-Schuler-Christian disease with xanthomatous orbital deposits and loss of membranous orbital bone may produce unilateral or bilateral exophthalmos. Congenital cysts and tumours such as teratoma epidermoid and dermoid, frequently developing from the suture lines or fissures, may extend into the orbit producing exophthalmos.

Excessive ossification of the bones of the orbit, as in the Albright-McCune-Sternberg syndrome, also termed polyostotic fibrous dysplasia (osteitis fibrosa cystica) may have exophthalmos as a symptom. Frequently craniostenosis, because of the shallow orbit not allowing room for the normal orbital contents, shows a type of pseudo-exophthalmos. This is seen particularly in oxycephaly, acrocephalo-syndactyly, dysostosis craniofacialis, and has been reported in hypertelorism, craniocleido-dysostosis and the syndrome described by Biemond.

Certain types of vascular anomalies in the orbit must also be considered congenital. Types of angiomas are of this nature.

These varied conditions if treated require surgery usually undertaken by the neurosurgeon, unless the lesion is located with reasonable certainty entirely in the orbit.

**Inflammation (infection)**

Inflammation is a frequent cause of exophthalmos, usually produced by infection of various types. Non-infectious oedema may also produce exophthalmos as found in venostasis, angioneurotic oedema, allergy and certain toxic poisons.

The inflammation may be primarily in the orbit; secondary, extending from adjacent structures; or it may result from arterial, venous and lymphatic metastases.

Inflammation of the orbit may involve the various tissues in cellulitis, thrombo-phlebitis, periostitis, tenonitis and myositis. The acute pyogenic infections may continue to abscess formation. Early treatment is directed toward removing the cause and instituting antibiotic medication.

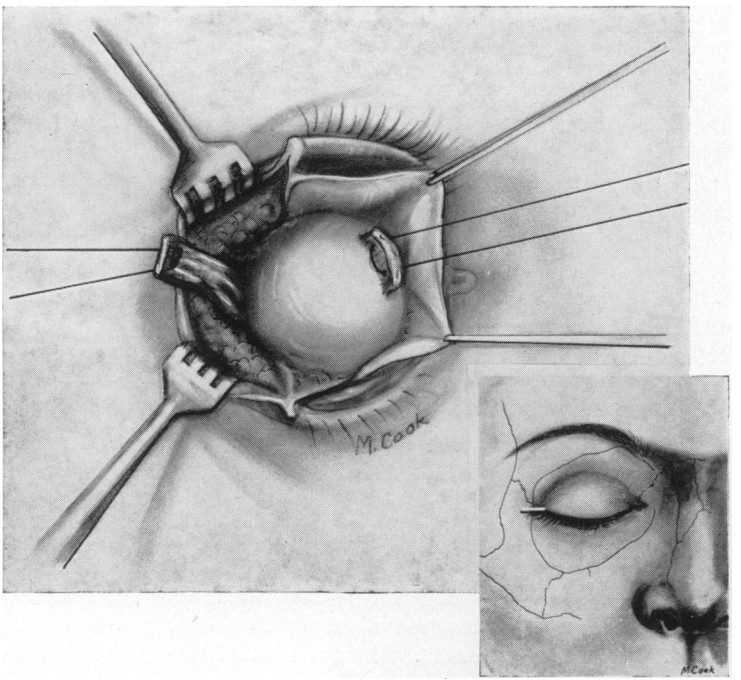


Fig. 1. Transconjunctival exposure with temporal canthotomy and incision through canthal ligament, septum orbitale and conjunctiva, retracting the globe nasally.

When an abscess has formed it should be opened. The incision is usually through the skin over the orbital rim, if possible, where there is most elevation, fluctuation and discolouration. The incision should only penetrate the skin and superficial tissues so as not to damage the muscles, nerves and blood vessels. Care must be taken not to make a broad horizontal incision through the upper lid for fear of damaging the levator muscle. A closed mouse tooth haemostat is then inserted into the wound to the depth of the abscess; the blades partly separated and withdrawn with the hopes of tapping and draining the pus. If pus is found, gauze packing will continue the drainage.

If the abscess is present within the muscle cone it may be necessary to approach it through a transconjunctival route (which will be mentioned later) (Fig. 1). It is not good practice to incise and jab around the orbit

blindly seeking pockets of pus when signs do not point to an abscessed area. However, if it seems necessary an 18-gauge needle might be introduced through a skin incision in the lower outer quadrant over the orbital rim, as one does when injecting retrobulbar anaesthesia, with the hopes of locating and aspirating pus. Usually this procedure should be condemned.

Chronic infections such as syphilis, tuberculosis and rarely sarcoid may produce granulomas involving the tissues of the orbit producing exophthalmos. Parasitic and mycotic infections are rare. These tumours are removed when necessary according to location; to be discussed later.

Pseudo-tumour is the most frequent condition requiring differentiation from true tumour, especially in the adult of middle age. The frequency of pseudo-tumour of the orbit is high on the list of retrobulbar space-consuming lesions producing exophthalmos. It is believed to be of a type of chronic granulation, probably due to low grade infection. It may be induced by intraorbital foreign body, blood clot, or one of the chronic granulomata.

Many of the symptoms are similar to those of true tumour. However, because of its inflammatory nature, the pseudo-tumour may form more rapidly, may be bilateral, may produce oedema of the lids, chemosis of the conjunctiva, and may be somewhat painful—symptoms rarely found in the unilateral, non-inflammatory slower growing neoplasm.

Unless easily removed, which usually is not possible because of its infiltrative nature, biopsy material is taken for diagnosis. Pseudo-tumour must also be differentiated from the so-called thyrotropic or ophthalmoplegic exophthalmos, as the appearance, symptoms and particularly tissue pathology may be quite similar. It is also similar to chronic orbital myositis described by Francois (1950) and his co-workers. Pseudo-tumour tissue examination reveals oedema of connective tissue and muscles which undergo fibrous and hyaline degeneration with localized foci of round cells, mononuclear leucocytes and lymphocytes, usually surrounding occluded vessels, a condition closely resembling that found in thyrotropic exophthalmos.

If pseudo-tumour is diagnosed, further surgery is contraindicated, the wound is closed, and temporising is in order. Meticortone or meticortelone may be used. Antibiotics may be of advantage. If lymphocytic involvement is marked, radiation may help. Regression is the rule. Surgery, if attempted, must needs be radical because of the infiltrative nature of the pseudo-tumour. It would produce irreparable damage to function and should not be resorted to.

Cavernous sinus thrombosis produces symptoms of inflammatory exophthalmos with oedema, venous engorgement, tenderness and pain in the orbit and adjacent structures. Vision is usually impaired with papilloedema, optic neuritis and congestion of retinal veins.

Symptoms of local, cerebral and systemic infection of toxic and pyaemic nature are usually present. The thrombosis spreads by way of the circular sinus to the opposite side.

Antibiotic drugs instigated early may be of great aid in recovery. Anticoagulants (heparin and dicumerol) and specific sera have been employed to advantage. Surgical treatment, if resorted to, is in the hands of the neurosurgeon.

**Vascular**

Engorgement or dilatation of blood vessels, venous, arterial or undifferentiated, in the orbit or in structures draining the orbit (i.e., cavernous sinus), will add to its bulk and produce exophthalmos. This condition is found in orbital varix, aneurysm and arterio-venous fistula, as well as various types of haemangiomas in which there is vascular proliferation. Vascular occlusion by embolus and thrombosis as mentioned above, may produce oedema and congestion leading to exophthalmos.

Vascular lesions may produce characteristic symptoms. Discolouration may be in evidence if the vessels are near the surface. Fluctuation on orbital pressure may be found. The amount of exophthalmos may vary with lowering the head and with stress and strain. Pulsation may be present with venous involvement which may be increased by pressure on the jugular vein. It is present in arteriovenous shunt when bruit also may be elicited with head noises and reduced by pressure upon the carotid artery in the neck. This must be differentiated from cerebral herniation through dehiscent orbital wall which may also have pulsating exophthalmos. In both venous and arterial lesions the globe is reducible and the ocular motility usually remains intact.

Radiologic studies frequently show calcified plaques in the area of the lesion. Angiography may reveal further evidence.

Consultation with the neurologist or neurosurgeon to determine extent and location of the lesion is imperative. If the lesion is localised in the orbit and is slowly growing and cosmetically unsightly, X-ray may be tried in younger patients but there is some risk of damage to vision. Injection of a sclerosing substance, if it can be confined to an accessible area, may be considered. If surgery is indicated, the approach is determined, depending upon location. If the lesion shows intracranial symptoms or if there is danger of uncontrolled bleeding, a neurosurgeon should be consulted.

Haemangioma, a very frequent cause of exophthalmos, will be discussed under neoplasm (tumour).

**Trauma**

Trauma, or other conditions conducive to bleeding, such as scurvy, various types of vascular diseases and blood dyscrasias may produce an intraorbital haemorrhage, which, because of its bulk, results in exoph-

thalmos. This haemorrhage may absorb, may form an organised clot developing into solid fibrotic tumour, or may increase in growth rapidly or slowly, as a haematic tumour or cyst, occasionally termed hygroma. The fibrous wall contains granulation tissue, round cells and products of mesodermal degeneration (lipoids, cholesterol, etc.) with cavity containing degenerated blood. These tumours may occasionally have interim bleeding from vascular elements within the growth.

Exophthalmos is a common symptom. Discolouration of the skin from blood elements may be in evidence. Fluctuation may be elicited if cystic.

Exploration with biopsy needle may be considered proper if localisation is determined. Occasionally the tumour may require removal through the Krönlein or other methods of approach.

Fracture of the wall between orbit and nasal sinuses may introduce air into the orbit, which increases with blowing the nose and sneezing, with the production of exophthalmos. This orbital emphysema is diagnosed by crepitus upon palpation through the lids or soft orbital tissue. Consultation should be had with the rhinologist. Antibiotics should be given to prevent infection.

## METABOLIC EXOPHTHALMOS

### Endocrine

Probably the most debatable condition producing exophthalmos is that seen in connection with thyroid, thyroid-pituitary or polyglandular affections. The exophthalmos may be associated with the hyperthyroid state, may follow thyroid surgery or may occur in the hypothyroid or those with apparently normal metabolism, although a history of previous disturbances is the rule. The condition is bilateral although one side is frequently more involved.

Thyrotoxicosis may produce exophthalmos probably as the result of thyroid overactivity induced by sympathetic stimulation from an excess of thyroxin. The exophthalmos, frequently only relative, may be exaggerated by lid lag or retraction and is reducible upon pressure as there is no increase of solid retrobulbar bulk. Orbital surgery is not necessary although orbital decompression and shortening of the recti muscles have been suggested. Some cosmetic improvement can be afforded and the exposed eye can better be protected by a lateral tarrsorhaphy of 3 to 4 mm., thus narrowing the palpebral space and reducing the exophthalmos a slight amount.

Thyrotropic exophthalmos (exophthalmic ophthalmoplegia) probably due to anterior pituitary overaction, which may be aggravated by thyroid underaction, shows definite increase in retrobulbar bulk to account for the irreducible exophthalmos. The extra ocular muscles are oedematous with lymphocytic and fat infiltation, later fibrosis and degeneration which,

in addition to the increase in bulk, produces paresis and paralysis of the extra ocular muscles.

Many do not agree with the classification of the thyrotoxic and thyrotropic types, believing that one merges into the other and are the same condition varying in degree of severity. Others believe the endocrine pattern is different between the two types only in the combination of the entire glandular group as a whole.

Until more is known of the aetiology, the medical treatment can only be haphazard and usually ineffective. Therapeutic X-ray of the orbit, and pituitary have been tried with variable results reported. Treatment with thyroxin or radioactive iodine (131) may be tried (Ruedemann and Corrigan, 1953). Some cases reported show spontaneous arrest. Hypophysectomy has been tried in desperate cases. A recent report is of satisfactory recovery with regard to the exophthalmos, the patient being maintained on cortisone, thyroid and posterior pituitary medication. Thyroidectomy may be dangerous by removing a hormone counteracting the pituitary hormone which appears to influence production of exophthalmos.

Before the exophthalmos reaches the state of jeopardising vision by optic atrophy or by corneal ulceration from exposure, and paresis or paralysis of the extra ocular muscles, decompression should be resorted to. Tarsorrhaphy is usually not sufficient. The surgical treatment will be outlined in connection with removal of deep intraorbital tumours by the Berke modification of the Krönlein operation which gives the orbit room to expand into the temporal fossa through the opening produced by the removal of a large portion of the lateral orbital wall (Fig. 2).

**Lipodystrophies**

The most prominent of the lipodystrophies producing exophthalmos is diabetic exophthalmic dysostosis (Hand-Schuler-Christian disease) with deposits in the orbit and optic nerve sheaths of cholesterol and cholesterol esters, producing granulomata packed with xanthomatous cells. Although the orbital bone may be destroyed the xanthomatous membrane prevents cerebral herniation. Letterer-Siwe's disease in infants and eosinophilic granuloma in adolescent and young adults are probably the same affection in various forms and intensities. Gargoylism or dysostosis multiplex may also have exophthalmos as a symptom from lipoid deposits in the orbit.

No treatment is effective ; X-ray and radium have been tried.

Osteitis deformans may be classified under metabolic affections, as it results from hormonal disturbance, producing absorption of normal bone with hyperplastic replacement, which crowds the orbit and produces exophthalmos. Osteofibrosis cystica is a similar condition with the production of cysts and tumour masses. These conditions have been mentioned earlier.

## TUMOURS

Tumours of the orbit may be divided into primary, secondary extension from adjacent structures and metastatic from foci elsewhere.

Exophthalmos is the cardinal symptom of both the hidden and apparent tumours. If no exophthalmos exists there is no retrobulbar space consuming lesion. If present exophthalmos is usually the first and perhaps the only symptom for a considerable period of time.

The frequency of various types of primary tumours of the orbit are listed by Reese (1956), whose selection includes 90 per cent. of the entire group producing exophthalmos. They are with slight modification :

**Primary tumours (in order of frequency)**

1. ANGIOMA (haemangioma)
2. HAEMOPOIETIC-RETICULOSES (lymphoma, lymphosarcoma)
3. PSEUDO-TUMOUR chronic granulomata—not true tumours—discussed under inflammation
4. EPITHELIAL TUMOURS (lacrimal gland tumours)
5. NERVE TISSUE TUMOURS (neurofibroma, neurileminoma and glioma)
6. MESENCHYMAL TUMOURS (meningioma)
7. MUSCLE TUMOURS (very rare)
8. OSTEOMAS (very rare)

Most haematomas, dermoid cysts, neurofibroma and astrocytoma of the optic nerve occur in childhood and should be considered along with the developmental anomalies producing exophthalmos. One might bear in mind Sturge-Weber syndrome with cutaneous naevus over the trigeminal distribution, glaucoma and meningeal vascular abnormalities. The treatment of these tumours depends upon their type and character, determined by biopsy and also upon their location and size.

Although the successful treatment of haemangioma has been obtained from sclerosing solution, if small and accessible, and also by electrolysis and radiation, to which they are usually resistant, the best results are surgical removal by the most feasible approach. They usually shell out readily with very little bleeding.

Diagnosis of lymphoma (benign) and lymphosarcoma (malignant) is usually by biopsy and may be made from biopsy elsewhere such as lymph nodes, sternal marrow, etc.

The most satisfactory treatment is by radiation. Excision followed by radiation might be considered.

Developmental tumours such as dermoid should be removed surgically by the best approach.

Lacrimal gland tumours should be removed by surgery, radical when necessary. Biopsy is definitely needed as there are various types. If a pseudo-tumour is present one should temporise ; if lymphosarcoma (adenocystic type), irradiation may be used ; if of mixed type and encapsu-

lated, blunt dissection, including the entire capsule, may be proper. If found to be carcinomatous, the orbit should be exenterated forthwith together with involved or suspicious bone.

Nerve tissue tumours, either neurofibroma or glioma, should be removed entirely, usually necessitating sacrificing vision by including the optic nerve to the extent of its involvement. In these cases if the optic foramen shows dilatation on X-ray, or middle fossa symptoms are present, the transfrontal route is the correct approach in order to expose the intracranial extension.

Meningioma should be removed surgically by the transfrontal exposure as it most frequently, probably without exception, has intracranial involvement.

Proper treatment can be determined only after correct tissue diagnosis has been established. For this the tumour must first be located. From the symptoms, which should be carefully recorded, the location of a circumscribed and, with more difficulty, a diffuse infiltrative lesion may be revealed.

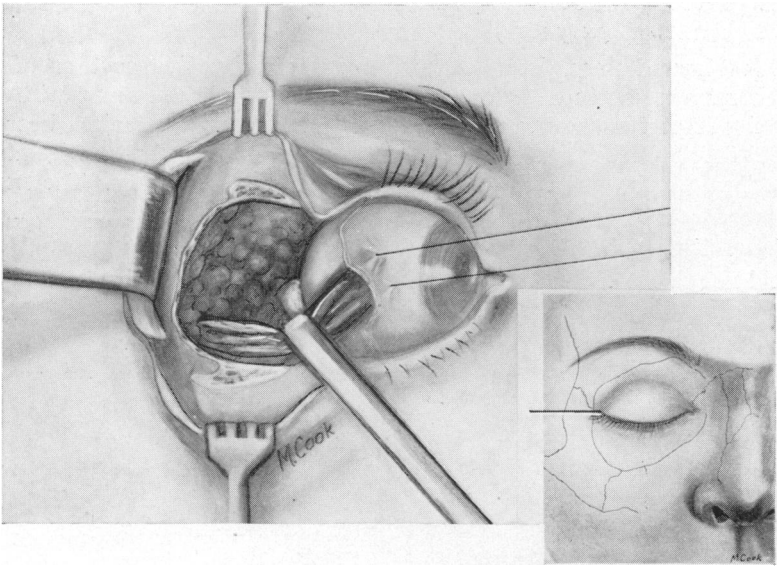


Fig. 2. Horizontal incision from temporal canthus exposing temporal fascia and muscle and lateral orbital wall which is sectioned and reflected laterally. Krönlein operation, Berke modification.

Reese (1951) emphasises the admonition that " unless a mass is palpable somewhere in the orbit no surgery should be attempted." He further states " I believe that no orbit should be explored, either for biopsy

purposes or for excision of any possible lesion unless the lesion has manifested itself as a mass which can serve as an object for surgical approach."

I would assume there is no objection to a transconjunctival approach for observation or digital palpation for a hidden retrobulbar mass too deep for surface localisation if:

1. The exophthalmos is progressing.
2. The vision is jeopardised from surface exposure of the globe or from optic atrophy.

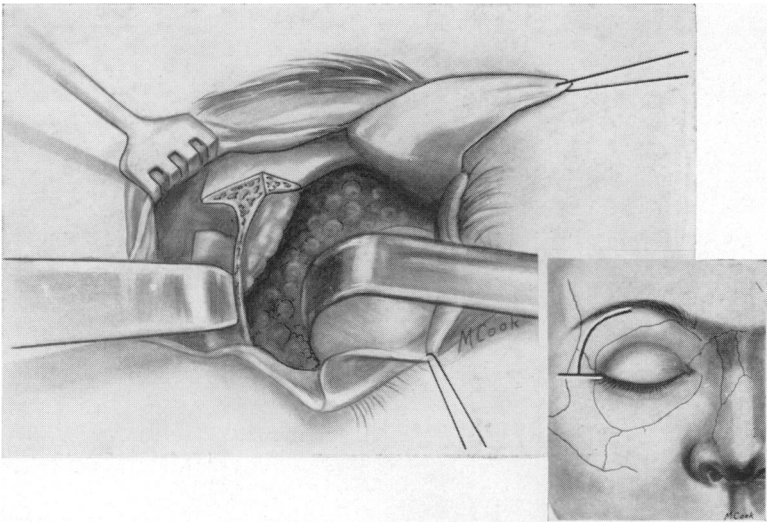


Fig. 3. Superior temporal exposure of orbital rim, which may be removed, and exposure of upper temporal quadrant of orbit by nasal reflection of the upper lid and downward retraction of the globe. Benedict operation, Reese modification.

3. The extraocular muscles are becoming paretic or paralytic.

When the lesion is exposed the next step is to establish the diagnosis through biopsy material or removal of the entire mass for microscopic study. If aspiration or biopsy are undertaken the material should be studied and reported immediately so that further operation, or closure, whichever is indicated, can terminate the procedure.

Possibly the best approach for diagnostic exposure, especially when the tumour is not definitely located, is the transconjunctival route via a temporal canthotomy with extension through the canthal ligament, and septum orbitale (Fig. 1). The conjunctiva of the fornix is incised around the temporal orbit to the vertical meridian. Retraction of the globe nasally allows for good lateral exposure for digital examination and affords an avenue for surgical procedure.

If after the transconjunctival exposure it is found necessary to open a wider and deeper field, the Krönlein-Berke operation (Fig. 2) (Berke, 1953) is an easy procedure by extending the horizontal incision laterally about 4 cm., exposing the lateral wall of the orbit and temporal fascia and muscle. With vertical retraction of the incision the lateral orbital rim is exposed and incision through the periosteum is made parallel with the rim. The periosteum externally and periorbitum internally are bluntly dissected from the bone. A section of the bone is removed from the lateral wall, preferably with a Stryker oscillating saw, thereby protecting the orbital contents, and the bone is reflected temporally to expose the lateral orbit contained in the periorbital membrane, for observation, palpation and surgical procedure.

Other more advantageous approaches may be made by incision elsewhere along the orbital rim. For lesions located superiorly and temporally, especially for lacrimal gland tumours, the Benedict (1945) exposure, modified by Reese (Fig. 3) (1951), which prevent damage to the

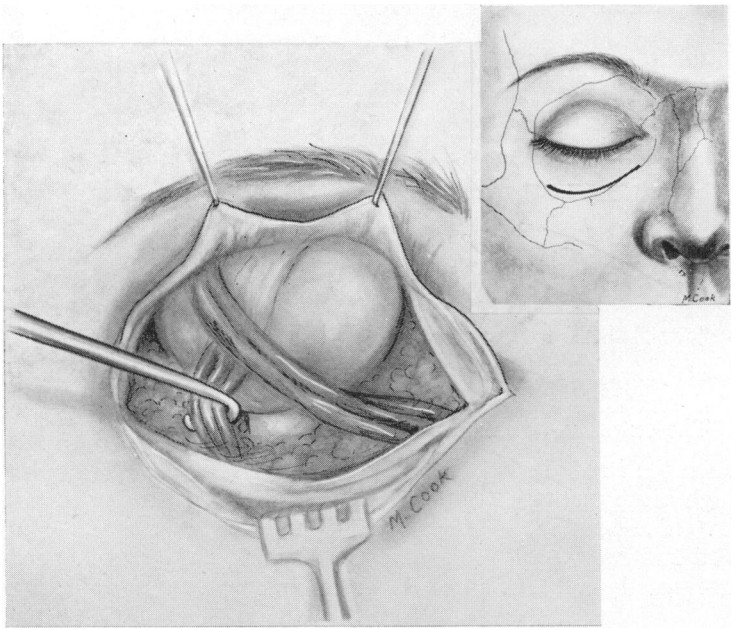


Fig. 4. Lower orbital exposure with incision at rim through skin, orbicular muscle and septum orbitale with upward retraction of the globe. Davis operation.

levator muscle is usually the operation of choice. A crescentic incision is made parallel with and about 5 mm. above the upper orbital rim, usually through the eyebrow, down to the periosteum which is bluntly dissected to the rim and into the orbit. The globe can be retracted down-

ward to expose the upper temporal quadrant. The upper lid can be retracted nasally.

For lesions located in the lower portion of the orbit the Davis (1940) (Fig. 4) incision is made just above the inferior orbital rim through skin, orbicular muscle and septum orbitale. With upward retraction of the globe the lower portion of the orbit can be exposed for inspection, palpation and surgery.

For lesions located in the upper nasal quadrant Callahan (1956) (Fig. 5) has devised an approach with an incision beginning at a point 1 cm. nasal to the inner canthus extending upward skirting the orbital rim to its highest point. The periosteum is incised over the rim, the trochlear attachment chipped away with chisel and periorbitum dissected from the bone to give access to the area. The trochlea is reattached before closing the wound.

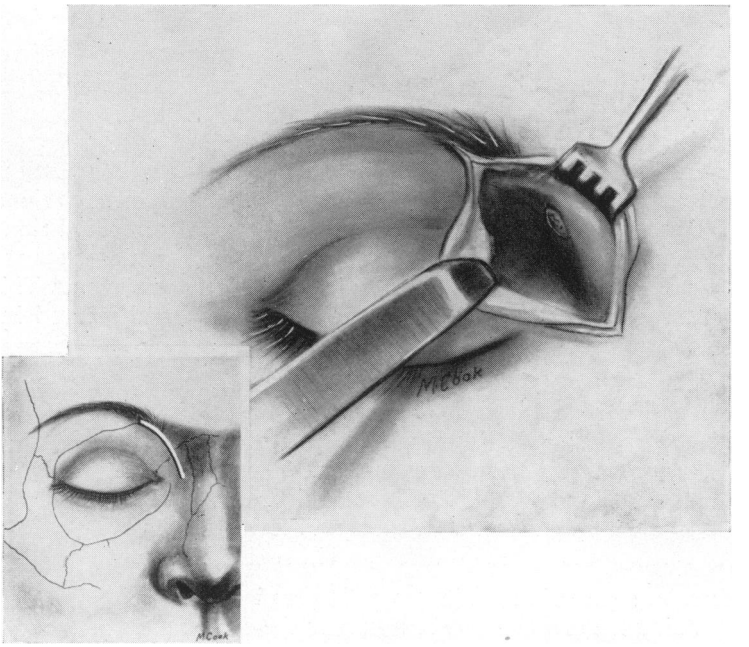

Fig. 5. Upper nasal exposure with incision beginning nasal to inner canthus extending to supra-orbital notch with exposure of orbital rim and temporary separation and reflection of the trochlea, the globe being retracted downward and temporally. Callahan operation.

In any of the operations, the extra-ocular muscles interfering with retraction and exposure may need to be severed and reflected from the field, to be reattached before the finish of the operation. Not infrequently

nerves and blood vessels must be sacrificed—often the optic nerve in glioma and neurofibroma. One should have permission from the parent or guardian, if the patient is not responsible.

Transcranial approach to the orbit by way of the Naffziger operation or modification should be used in lesions of the orbital apex, optic foramen and middle cranial fossa to include all cases of orbital extension from the cranial cavity or cranial extension from the orbit. In these instances X-rays of the bony orbit, optic foramen, orbital fissures and structures of the middle cranial fossa may be of great diagnostic importance. The surgical procedure is in the hands of the neurosurgeon. The most frequent tumours of this kind are the neurofibroma, glioma (astrocytoma) and retroblastoma in children and the meningioma in adults.

Whether the transfrontal Naffziger operation for decompression of the orbit in so-called malignant thyrotropic exophthalmos is the operation of choice remains a question for discussion. I am inclined to favour the lateral approach of Krönlein-Berke.

Orbital exenteration is advisable in orbital sarcoma, excepting lymphosarcoma when X-ray is beneficial. It may be indicated in diffuse mixed tumours of the lacrimal gland ; it should definitely be done in carcinoma of the lacrimal gland. It may be necessary in secondary involvement of the orbit from epithelioma of the lid. If the lids are to be spared in the orbital exenteration the incision is through the fornices of the conjunctiva. If the lids are to be included the incision is around the orbital rim, with blunt dissection, as much as possible, of the periorbitum throughout and cauterization of the nerves and vessels in the orbital apertures. A Thiersch graft transplant is inserted against the orbital wall and secured in firm contact.

Tarsorrhaphy may be used in exophthalmos for the protection of the cornea and for postoperative positioning of the eye. This is simply performed by closing the lids with two silk sutures inserted a few millimetres from the lid margins and tied over pegs. They should be placed so that they are lateral and medial to the corneal limbus (one on either side) so as not to irritate the cornea. If the eye is to be aligned these sutures are passed through the episcleral tissue on either side of the corneal limbus.

Secondary extension of inflammation and tumours from the tissues surrounding the orbit usually necessitates the cooperation of the ophthalmologist and rhinologist. These lesions extending from the nasal sinuses and sinus wall and from the pharynx may invade the orbit and produce exophthalmos and require surgical treatment for reduction of the orbital contents.

Metastatic tumours lodging in the orbit are not uncommon. The carcinoma has its most frequent primary lesions in the breast, uterus, kidney, thyroid, prostate and pancreas. The sarcoma and, rarely, malignant

melanoma may be found in the orbit. Orbital surgery may be undertaken if life expectancy warrants the procedure.

## CONCLUSION

Conditions producing exophthalmos are numerous and varied.

Proper treatment rests entirely upon the recognition of the type of condition producing the exophthalmos.

Although the treatment frequently should not be attempted by the ophthalmologist alone it is his responsibility to make the diagnosis with such aid as is necessary and see that proper treatment is instituted.

I know of no better way to close this Moynihan Lecture, which it has been my privilege to present, than to cite a phrase from Montaigne's Essays, often quoted by Lord Moynihan, as it most aptly applies to my humble effort this evening.

" I have gathered a posie of other men's flowers
And nothing but the thread that binds them is mine own."

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## REFERENCES

BENEDICT, W. L. (1945) *Amer. J. Ophthal.* **32,** 763-773.
BERKE, R. N. (1953) *Trans. Amer. ophthal. Soc.* **51,** 193-232.
CALLAHAN, A. (1956) *Surgery of the Eye*, p. 447. Springfield, Ill., Thomas.
DAVIS, F. A. (1940) *A.M.A. Arch. Ophthal.* **23,** 735-821, 957-1018.
DREWS, L. C. (1957) *Amer. J. Ophthal.* **43,** 37-57.
FRANCOIS, J., RABAEY, M., and EVENS, L. (1950) *Ophthalmologica (Basel)* **121,** 105-120,
HENDERSON, J. W. (1956) *A.M.A. Arch. Ophthal.* **56,** 678-684.
INGALLS, R. G. (1953) *Tumors of the Orbit*, p. 401. Springfield, Ill., Thomas.
REESE, A. B. (1951) *Tumors of the Eye*, p. 574. New York, Paul B. Hoeber.
———— (1956) *Amer. J. Ophthal.* **41,** 3-10.
RUEDEMAN, A. D., and CORRIGAN, R. E. (1953) *Trans. Amer. ophthal. Soc.* **51,** 333-344.

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## ANNUAL GENERAL MEETING AND SCIENTIFIC MEETING
## 11th December 1957

THE ANNUAL GENERAL MEETING of the College will be held on 11th December 1957, and it is hoped that as many Fellows and Members as possible will attend. The provisional programme is as follows :

| | |
|---|---|
| 10 a.m -2.30 p.m. | Scientific displays arranged by each of the College Departments. |
| 11.45 a.m.-12.45 p.m. and 2.00 p.m. - 2.30 p.m. | Programme of scientific films. |
| 2.30 p.m. | Bradshaw Lecture by Sir Russell Brock. |
| 4.00 p.m. | Annual Meeting of Fellows and Members. |
| 7.00 p.m. for 7.30 p.m. | Monthly Subscription Dinner—tickets 30s. each. |